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Search Results -

Term	Documents
LACTOGEN	943
LACTOGENS	76
(LACTOGEN AND 10).USPT,PGPB,JPAB,EPAB,DWPI,TDBD.	28
(L10 AND LACTOGEN).USPT,PGPB,JPAB,EPAB,DWPI,TDBD.	28

Database:
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EPO Abstracts Database
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IBM Technical Disclosure Bulletins

Search: L11

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Search History

DATE: Monday, November 17, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=ADJ			
L11	L10 and lactogen	28	L11
L10	L9 and interferon	65	L10
L9	hpl	1009	L9
L8	isplp	2	L8
L7	peyman-john-a.in.	16	L7
L6	interferon and supress$3 and placent$3 and lactogen	15	L6
L5	interferon and supress$3 placent$3	0	L5
L4	interferon and supress$3	571	L4
L3	interferon and supress$3 placent$3 and lactogen	0	L3
L2	interferon and supress$3 placent$3 and lactogen and peptide	0	L2
L1	interferon-supressing placental lactogen peptide	0	L1

END OF SEARCH HISTORY

```
          ********** STN Columbus **********

FILE 'MEDLINE'
FILE 'JAPIO'
FILE 'BIOSIS'
FILE 'SCISEARCH'
FILE 'WPIDS'

-> s interferon and supressing and placental and lactogen and peptide
L1          0 INTERFERON AND SUPRESSING AND PLACENTAL
AND LACTOGEN AND PEPTIDE

-> s interferon-supressing
L2          0 INTERFERON-SUPRESSING

=> s interferon supressing
L3          0 INTERFERON SUPRESSING

=> s interferon suppressing
  1 FILES SEARCHED...
L4          9 INTERFERON SUPPRESSING

-> s interferon and suppressing and placental and lactogen and peptide
L5          2 INTERFERON AND SUPPRESSING AND PLACENTAL
AND LACTOGEN AND PEPTID
            E

-> isplp
L6          2 ISPLP

  > s isplp
L7          2 ISPLP

=> l7 and interferon
L8          2 L7 AND INTERFERON

  > s interferon and inhibiting
L9       8203 INTERFERON AND INHIBITING

=> l9 and lactogen
L10         1 L9 AND LACTOGEN

  > dup rem l10
PROCESSING COMPLETED FOR L10
L11         9 DUP REM L10 (2 DUPLICATES REMOVED)

=> dup rem l8
PROCESSING COMPLETED FOR L8
L12         1 DUP REM L8 (1 DUPLICATE REMOVED)

-> dup rem l7
PROCESSING COMPLETED FOR L7
L13         1 DUP REM L7 (1 DUPLICATE REMOVED)

-> dup rem l5
PROCESSING COMPLETED FOR L5
L14         1 DUP REM L5 (1 DUPLICATE REMOVED)

-> dup rem l4
PROCESSING COMPLETED FOR L4
L15         4 DUP REM L4 (5 DUPLICATES REMOVED)

-> d ibib abs l15 1-4

L15 ANSWER 1 OF 4 WPIDS COPYRIGHT 2003 THOMSON
DERWENT on STN DUPLICATE 1
ACCESSION NUMBER:    2002-424996 [45]  WPIDS
DOC. NO. CPI:        C2002-120339
TITLE:               ***Interferon*** - ***suppressing*** placental
                     lactogen peptides, useful for treating autoimmune
                     disease, inflammatory disease or organ transplant
                     rejection, e.g. lupus erythematosus, Crohn's disease,
                     eczema, septic shock or ischemia.
DERWENT CLASS:       B04
INVENTOR(S):         PEYMAN, J A
PATENT ASSIGNEE(S):  (PEYM-I) PEYMAN J A
COUNTRY COUNT:       1
PATENT INFORMATION:

   PATENT NO  KIND DATE    WEEK    LA  PG
   ---------------------------------------
   US 2002032154 A1 20020314 (200245)*     31

APPLICATION DETAILS:

   PATENT NO  KIND          APPLICATION     DATE
   ---------------------------------------------
   US 2002032154 A1 Provisional     US 2000-210082P 20000607
                    US 2001-876478  20010607


PRIORITY APPLN. INFO: US 2000-210082P 20000607; US
2001-876478
                20010607
AN  2002-424996 [45]  WPIDS
AB  US2002032154 A UPAB: 20020717
   NOVELTY - ***Interferon*** - ***suppressing*** placental
lactogen
   peptides which suppress IFN-gamma-stimulated expression of MHC
class II
   antigens, MHC class I antigens and/or ICAM-1 antigen, are new.
      DETAILED DESCRIPTION - An ***interferon*** -
***suppressing***
   placental lactogen peptide (which suppresses IFN-gamma-stimulated
   expression of MHC class II antigens MHC class I antigens and/or
   antigen) comprises:
      (a) N-terminal 28 residues of hPL, or hPL-1;
      (b) a 28-amino acid sequence with substantial identity to hPL or
   hPL-1, containing one or more conservative amino acid substitutions;
      (c) a derivative of hPL or hPL-1, having residues 5-27; or
      (d) a derivative of (b) having residues 5-27.
      The ***interferon*** - ***suppressing*** placental lactogen
   peptides (ISPLP) hPL and hPL-1 have the sequences S1 and S2
respectively.
      INDEPENDENT CLAIMS are also included for:
      (1) treating a human subject by administering to the subject a cell
   or tissue that has been treated with the peptide; and
      (2) treating a human subject by administering the ISPLP.

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al
a-
   His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al
a-
   His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
      ACTIVITY - Immunosuppressive; Antiinflammatory;
Neuroprotective;
   Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
      No biological data given.
      MECHANISM OF ACTION - Interferon gamma suppressor.
      USE - The peptide is useful for treating autoimmune disease,
   inflammatory disease or organ transplant rejection (claimed). These
   diseases include lupus erythematosus, multiple sclerosis, Crohn's
disease,
   asthma, hay fever, eczema, sepsis, septic shock or ischemia.
   Dwg.0/11

L15 ANSWER 2 OF 4    MEDLINE on STN              DUPLICATE 2
ACCESSION NUMBER:    96207396    MEDLINE
DOCUMENT NUMBER:     96207396    PubMed ID: 8615043
TITLE:               NF-kappaB activation Is delayed in mouse L929 cells
                     infected with ***interferon*** ***suppressing*** ,
                     but not inducing, vesicular stomatitis virus strains.
AUTHOR:              Boulares A H; Ferran M C; Lucas-Lenard J
CORPORATE SOURCE:    Molecular and Cell Biology Department,
University of
                     Connecticut, Storrs, 06269-3125, USA.
SOURCE:              VIROLOGY, (1996 Apr 1) 218 (1) 71-80.
                     Journal code: 0110674. ISSN: 0042-6822.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199606
ENTRY DATE:          Entered STN: 19960613
                     Last Updated on STN: 19980206
                     Entered Medline: 19960605
AB  Vesicular stomatitis virus (VSV) mutant T1026R1 of the Indiana (IN)
   serotype is a good inducer of interferon (IFN). This mutant was used to
   study the activation of NF-kappaB, a transcription factor necessary for
   IFN induction, in mouse L929 cells that were stably transfected with a
   chimeric gene containing the human IFN-beta gene promoter attached
to the
   chloramphenicol acetyltransferase (CAT) coding sequence. NF-kappaB
DNA
   binding activity was detected as early as 30 min after virus adsorption in
   nuclear extracts, increased up to 4 hr, and then remained constant for at
   least 6 additional hr. The kinetics of CAT expression correlated with
the
   kinetics of NF-kappaB nuclear DNA binding activity. Virus entry and
   delivery of viral components into the cytoplasm were required for
   NF-kappaB activation. Exposure of T1026R1 to one hit of UV
irradiation
   nearly completely reduced NF-kappaB activation. In cells infected with
   wild-type (wt) VSV (IN), a noninducer of IFN, NF-kappaB DNA
binding
   activity in the nucleus was delayed for several hours after virus
   adsorption. Coinfection of wt VSV and T1026R1 resulted in the
reduction
   of T1026R1-promoted NF-kappaB activation. This inhibitory activity
of wt
   VSV was abolished by one hit of UV irradiation. Under similar
conditions
   expression of the CAT gene was more UV resistant, suggesting that
IFN gene
   expression is regulated at multiple levels.

L15 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL
ABSTRACTS INC. on STN
ACCESSION NUMBER:    1990:4193 BIOSIS
DOCUMENT NUMBER:     PREV199089004193; BA89:4193
TITLE:               INTERFERON INDUCTION BY VIRUSES XVIII.
VESICULAR STOMATITIS
                     VIRUS NEW JERSEY A SINGLE INFECTIOUS
PARTICLE CAN BOTH
                     INDUCE AND SUPPRESS INTERFERON PRODUCTION.
AUTHOR(S):           GACCIONE C [Reprint author]; MARCUS P I
CORPORATE SOURCE:    DEP MOL CELL BIOL, UNIV
CONNECTICUT, STORRS, CONN
                     06269-3044, USA
SOURCE:              Journal of Interferon Research, (1989) Vol. 9, No. 5,
pp.
                     603-614.
                     CODEN: JIREDJ. ISSN: 0197-8357.
DOCUMENT TYPE:       Article
FILE SEGMENT:        BA
LANGUAGE:            ENGLISH
ENTRY DATE:          Entered STN: 5 Dec 1989
                     Last Updated on STN: 5 Dec 1989
AB  In contrast to wild-type vesicular stomatitis virus (VSV) of Indiana
   (Ind ) origin which express interferon (IFN) inducing- and IFN
   individuals particles of wild-type VSV of the New Jersey (N.J.)
serotype
   (Hazelhurst [H] isolate) paradoxically can both induce IFN and
suppress
   its induction in cells coinfected with a potent inducer of IFN. The
   properties of IFN induction, and its suppression, appear to reside in the
   particle that manifests infectivity. Analyses of IFN induction
   dose-response curves to measure IFN-inducing particles (IFP), and IFN
   yield-reduction curves to measure IFN induction-suppressing particles
   (ISP) generated by VSV-N.J.(H) in aged chick embryo cells revealed
that
   (i) a single particle per cell sufficed to induce a quantum (full) yield
   of IFN, or to suppress fully IFN production by a coinfecting inducing
   virus, and (ii) the addition of one or more IFP per cell did not suppress
   the yield of IFN beyond the plateau level. The time-course of IFN
   production in chick cells infected with VSV-N.J.(H) revealed about a
4-h
   lag, even when the cells were coinfected with a potent inducer that
   normally induced IFN 1 or 2 h sooner. Thus, VSV-N.J.(H) appears to
   regulate the production of IFN in cells-even that initiated by other
   inducers. Expression of IFP and ISP activities both required primary
   transcription, with respective genomic targets similar to those reported
   for VSV-Ind. N.J.(H) is the first wild-type VSV observed to express
IFP
   and ISP activities concomitantly. A model is presented to suggest how
   these two antagonistic properties might be expressed by a single
   infectious particle.

L15 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL
ABSTRACTS INC. on STN
ACCESSION NUMBER:    1989:353051 BIOSIS
DOCUMENT NUMBER:     PREV198988045165; BA88:45165
TITLE:               INFLUENCE OF INTERFERON CIRCULATING IN
THE BLOOD OF INTACT
                     GUINEA-PIGS ON THE FORMATION OF AN
IMMEDIATE ALLERGY.
AUTHOR(S):           ERMEKOVA D M [Reprint author]; ASPETOV R D;
ABDRASILOVA G S
CORPORATE SOURCE:    RES INST EPIDEMIOL MICROBIOL
INFECT DIS, MINIST HEALTH KAZ
                     SSR, ALMA-ATA, USSR
SOURCE:              Immunologiya, (1988) No. 6, pp. 57-58.
                     ISSN: 0206-4952.
DOCUMENT TYPE:       Article
FILE SEGMENT:        BA
LANGUAGE:            RUSSIAN
ENTRY DATE:          Entered STN: 2 Aug 1989
                     Last Updated on STN: 2 Aug 1989
AB  The data are presented on interferon detection in the blood serum of
   intact guinea pigs and influence of .gamma.-interferon on formation fo
   immediate allergy to Artemisia scoparia pollen. It has been found that
   the circulating blood of a great number of guinea pigs contains
   .gamma.-
   ***interferon*** ***suppressing*** the process of formation of
   immediate response to Artemisia scoparia pollen allergens.


=> d ibib abs l14

L14 ANSWER 1 OF 1 WPIDS COPYRIGHT 2003 THOMSON
DERWENT on STN DUPLICATE 1
ACCESSION NUMBER:    2002-424996 [45]  WPIDS
DOC. NO. CPI:        C2002-120339
TITLE:               ***Interferon*** - ***suppressing***
                     ***placental*** ***lactogen*** ***peptides*** ,
                     useful for treating autoimmune disease, inflammatory
                     disease or organ transplant rejection, e.g. lupus
                     erythematosus, Crohn's disease, eczema, septic shock or
                     ischemia.
DERWENT CLASS:       B04
INVENTOR(S):         PEYMAN, J A
PATENT ASSIGNEE(S):  (PEYM-I) PEYMAN J A
COUNTRY COUNT:       1
PATENT INFORMATION:

   PATENT NO  KIND DATE    WEEK    LA  PG
   ---------------------------------------
   US 2002032154 A1 20020314 (200245)*     31

APPLICATION DETAILS:

   PATENT NO  KIND          APPLICATION     DATE
   ---------------------------------------------
   US 2002032154 A1 Provisional     US 2000-210082P 20000607
                    US 2001-876478  20010607


PRIORITY APPLN. INFO: US 2000-210082P 20000607; US
2001-876478
                20010607
AN  2002-424996 [45]  WPIDS
AB  US2002032154 A UPAB: 20020717
   NOVELTY - ***Interferon*** - ***suppressing***
***placental***
   ***lactogen*** ***peptides*** which suppress
IFN-gamma-stimulated
   expression of MHC class II antigens, MHC class I antigens and/or
ICAM-1
   antigen, are new.
      DETAILED DESCRIPTION - An ***interferon*** -
***suppressing***
   ***placental*** ***lactogen*** ***peptide*** (which
suppresses
   IFN-gamma-stimulated expression of MHC class II antigens, MHC
class I
   antigens and/or ICAM-1 antigen) comprises:
      (a) N-terminal 28 residues of hPL, or hPL-1;
```

hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.
The ***interferon*** - ***suppressing*** ***placental*** ***lactogen*** ***peptides*** (ISPLP) hPL and hPL-1 have the sequences S1 and S2 respectively.
INDEPENDENT CLAIMS are also included for:
(1) treating a human subject by administering to the subject a cell or tissue that has been treated with the ***peptide*** ; and
(2) treating a human subject by administering the ISPLP.

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al a-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al a-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
ACTIVITY - Immunosuppressive; Antiinflammatory; Neuroprotective;
Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
No biological data given.
MECHANISM OF ACTION - ***Interferon*** gamma suppressor.
USE - The ***peptide*** is useful for treating autoimmune disease, inflammatory disease or organ transplant rejection (claimed). These diseases include lupus erythematosus, multiple sclerosis, Crohn's disease, asthma, hay fever, eczema, sepsis, septic shock or ischemia.
Dwg.0/11

> d ibib abs l13

L13 ANSWER 1 OF 1 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 1
ACCESSION NUMBER: 2002-424996 [45] WPIDS
DOC. NO. CPI: C2002-120339
TITLE: Interferon-suppressing placental lactogen peptides, useful for treating autoimmune disease, inflammatory disease or organ transplant rejection, e.g. lupus erythematosus, Crohn's disease, eczema, septic shock or ischemia.
DERWENT CLASS: B04
INVENTOR(S): PEYMAN, J A
PATENT ASSIGNEE(S): (PEYM-I) PEYMAN J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002032154	A1	20020314 (200245)*			31

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002032154	A1 Provisional	US 2000-210082P	20000607
		US 2001-876478	20010607

PRIORITY APPLN. INFO: US 2000-210082P 20000607; US 2001-876478 20010607
AN 2002-424996 [45] WPIDS
AB US2002032154 A UPAB: 20020717
NOVELTY - Interferon-suppressing placental lactogen peptides which suppress IFN-gamma-stimulated expression of MHC class II antigens, MHC class I antigens and/or ICAM-1 antigen, are new.
DETAILED DESCRIPTION - An interferon-suppressing placental lactogen peptide (which suppresses IFN-gamma-stimulated expression of MHC class II antigens, MHC class I antigens and/or ICAM-1 antigen) comprises:
(a) N-terminal 28 residues of hPL, or hPL-1;
(b) a 28-amino acid sequence with substantial identity to hPL or hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.
The interferon-suppressing placental lactogen peptides (***ISPLP***) hPL and hPL-1 have the sequences S1 and S2 respectively.
INDEPENDENT CLAIMS are also included for:
(1) treating a human subject by administering to the subject a cell or tissue that has been treated with the peptide; and
(2) treating a human subject by administering the ***ISPLP*** .

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al a-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al a-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
ACTIVITY - Immunosuppressive; Antiinflammatory; Neuroprotective;
Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
No biological data given.
MECHANISM OF ACTION - Interferon gamma suppressor.
USE - The peptide is useful for treating autoimmune disease, inflammatory disease or organ transplant rejection (claimed). These diseases include lupus erythematosus, multiple sclerosis, Crohn's disease, asthma, hay fever, eczema, sepsis, septic shock or ischemia.
Dwg.0/11

L12 ANSWER 1 OF 1 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 1
ACCESSION NUMBER: 2002-424996 [45] WPIDS
DOC. NO. CPI: C2002-120339
TITLE: ***Interferon*** -suppressing placental lactogen peptides, useful for treating autoimmune disease, inflammatory disease or organ transplant rejection, e.g. lupus erythematosus, Crohn's disease, eczema, septic shock or ischemia.
DERWENT CLASS: B04
INVENTOR(S): PEYMAN, J A
PATENT ASSIGNEE(S): (PEYM-I) PEYMAN J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002032154	A1	20020314 (200245)*			31

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002032154	A1 Provisional	US 2000-210082P	20000607
		US 2001-876478	20010607

PRIORITY APPLN. INFO: US 2000-210082P 20000607; US 2001-876478 20010607
AN 2002-424996 [45] WPIDS
AB US2002032154 A UPAB: 20020717
NOVELTY - ***Interferon*** -suppressing placental lactogen peptides which suppress IFN-gamma-stimulated expression of MHC class II antigens, MHC class I antigens and/or ICAM-1 antigen, are new.
DETAILED DESCRIPTION - An ***interferon*** -suppressing placental lactogen peptide (which suppresses IFN-gamma-stimulated expression of MHC class II antigens, MHC class I antigens and/or ICAM-1 antigen) comprises:
(a) N-terminal 28 residues of hPL, or hPL-1;
(b) a 28-amino acid sequence with substantial identity to hPL or hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.
The ***interferon*** -suppressing placental lactogen peptides (***ISPLP***) hPL and hPL-1 have the sequences S1 and S2 respectively.
INDEPENDENT CLAIMS are also included for:
(1) treating a human subject by administering to the subject a cell or tissue that has been treated with the peptide; and
(2) treating a human subject by administering the ***ISPLP*** .

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al a-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al a-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
ACTIVITY - Immunosuppressive; Antiinflammatory; Neuroprotective;
Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
No biological data given.
MECHANISM OF ACTION - ***Interferon*** gamma suppressor.
USE - The peptide is useful for treating autoimmune disease, inflammatory disease or organ transplant rejection (claimed). These diseases include lupus erythematosus, multiple sclerosis, Crohn's disease, asthma, hay fever, eczema, sepsis, septic shock or ischemia.
Dwg.0/11

=> d ibib abs l11 1-9

L11 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:796985 CAPLUS
DOCUMENT NUMBER: 139:287284
TITLE: Solution phase hybridization-based methods for detecting and quantitating nucleic acid analytes
INVENTOR(S): Stephan, Jean-Philippe F.; Tsai, Siao Ping; Wong, Wai Lee Tan; Billeci, Todd
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 94 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003083440	A2	20031009	WO 2003-US9726	20030328

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2002-368669P P 20020329
AB The present invention provides novel soln. phase hybridization-based methods for detecting and quantitating nucleic acid analytes. Methods comprising use of novel capture polymers and/or signaling systems are provided. Use of these novel capture polymers and/or signaling systems provides significant improvements in signal to noise ratio, specificity, sensitivity and ease of development and use as compared to existing soln. phase nucleic acid detection and quantitation methods. The invention further provides compns., kits and articles of manuf. for practicing methods of the present invention.

L11 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:390758 CAPLUS
DOCUMENT NUMBER: 138:390934
TITLE: Topical and transdermal administration of peptidyl drugs with hydroxide-releasing agents as skin permeation enhancers
INVENTOR(S): Luo, Eric C.; Jacobson, Eric C.; Hsu, Tsung-Min
PATENT ASSIGNEE(S): Dermatrends, Inc., USA
SOURCE: U.S., 13 pp., Cont.-in-part of U.S. Ser. No. 569,889.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 23
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6565879	B1	20030520	US 2000-687937	20001013
US 2001038862	A1	20011108	US 2000-737831	20001214
US 6558695	B2	20030506		
US 2003147943	A1	20030807	US 2003-349582	20030122

PRIORITY APPLN. INFO.: US 1999-465098 A2 19991216
US 2000-569889 A2 20000511
US 2000-687937 A2 20001013
US 2000-737831 A3 20001214
AB A method is provided for increasing the permeability of skin or mucosal tissue to a topically or transdermally administered pharmacol. or cosmeceutically active peptide, polypeptide or protein. The method involves use of a specified amt. of a hydroxide-releasing agent, the amt. optimized to increase the flux of the peptide, polypeptide or protein through a body surface while minimizing the likelihood of skin damage, irritation or sensitization. Formulations and drug delivery devices employing hydroxide-releasing agents as permeation enhancers are provided as well. An in vitro human cadaver skin permeation study was conducted using 0.18% leuprolide soln. and 3.6% sodium hydroxide.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:332611 CAPLUS
DOCUMENT NUMBER: 136:337369
TITLE: Real time quantitative aptamer-PCR or immuno-PCR with detectable non-primer probes
INVENTOR(S): Dodge, Anthony H.; Meng, Yu-ju G.; Sims, Paul W.; Sinicropi, Dominick V.; Williams, P. Mickey; Wong, Wai Lee
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 37 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002051974	A1	20020502	US 1999-449204	19991124

PRIORITY APPLN. INFO.: US 1998-110259P P 19981130
AB The invention relates to a method for a novel method for detecting the presence of a target compd., in a sample which may contain the target compd., using a nucleic acid detector mol., amplification and quantitation or detection of the detector mol. The method uses the following steps: (a) exposing a sample, which may contain or is suspected of contg. the target compd., to a capture mol. capable of binding to the target mol. to form a capture mol.: target mol. complex; (b) adding to the capture mol.: target mol. complex, a detector mol. contg. a nucleic acid moiety and capable of specifically binding to the target mol.; and (c) amplifying the nucleic acid moiety by PCR amplification, and (d) quantitating or detecting the PCR amplified nucleic acid moiety using a detectable non-primer probe capable of binding to the nucleic acid moiety. In the method of the invention, the capture mol. may be an antibody, phage antibody, aptamer or other receptor or binding partner for the analyte of interest. The detector mol. may be either a nucleic acid labeled antibody

achieved by detecting the amplified nucleic acid (nucleic acid moiety on the labeled antibody or aptamer) with a detectable non-primer probe capable of binding to the amplified nucleic acid, preferably in real time. The invention, therefore, provides improvements in quantitation and sensitivity over immuno-PCR and ELONA assays which have been used for
protein analytes, by utilizing a PCR amplification and quantification technic used only for application to the real time detection of nucleic acids. The invention also provides improvements over conventional ELISA
assays in sensitivity. The use of non-primer probes, preferably with real time anal., e.g., the TaqMang system, in an aptamer-PCR or an immuno-PCR
assay as in the invention, overcomes the shortcomings of prior art processes discuss above. Since the PCR reaction products are not subjected to post-PCR manipulations, the risk of product contamination in
assays is significantly lowered. Monitoring the PCR reaction in 'real time' allows the collection of data across many cycles (e.g. cycle 1-50) instead of at an endpoint PCR stage, as in conventional immuno-PCR (e.g.
cycle 25 only), therefore allowing for a greater range of detectable amplicon. The method of the invention can detect the target mol. at a concn. of less than 1.0x10-1-g/mL, generally about 1.0x10-15 to about 1.0x10-8 g/mL. Detecting vascular endothelial growth factor (VEGF) using
the aptamer rt-PCR assay of the invention is described.

L11 ANSWER 4 OF 9 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 1
ACCESSION NUMBER: 2002-082332 [11] WPIDS
CROSS REFERENCE: 2001-441621 [47]; 2001-488538 [53]; 2002-065878 [09];
2002-074630 [10]; 2002-082296 [11]; 2002-105506 [14]; 2002-470580 [50]; 2003-341108 [32]; 2003-361827 [34]; 2003-370772 [35]; 2003-370773 [35]; 2003-456180 [43]; 2003-531377 [50]; 2003-554600 [52]; 2003-644797 [61]; 2003-658676 [62]; 2003-669970 [63]; 2003-670893 [63]; 2003-677996 [64]; 2003-720863 [68]; 2003-720864 [68]
DOC. NO. CPI: C2002-024825
TITLE: Composition useful for delivering peptidyl drugs comprises a hydroxide releasing agent.
DERWENT CLASS: B04 D16 P32 P34
INVENTOR(S): HSU, T; LUO, E C
PATENT ASSIGNEE(S): (HSUT-I) HSU T; (LUOE-I) LUO E C; (DERM-N) DERMATRENDS INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2001038862	A1	20011108	(200211)*		16
US 6558695	B2	20030506	(200338)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 2001038862	A1	CIP of	US 1999-465098	19991216
		CIP of	US 2000-569889	20000511
		CIP of	US 2000-687937	20001013
			US 2000-737831	20001214
US 6558695	B2	CIP of	US 1999-465098	19991216
		CIP of	US 2000-569889	20000511
		CIP of	US 2000-687937	20001013
			US 2000-737831	20001214

PRIORITY APPLN. INFO: US 2000-737831 20001214; US 1999-465098
19991216; US 2000-569889 20000511; US 2000-687937 20001013
AN 2002-082332 [11] WPIDS
CR 2001-441621 [47]; 2001-488538 [53]; 2002-065878 [09]; 2002-074630 [10];
2002-082296 [11]; 2002-105506 [14]; 2002-470580 [50]; 2003-341108 [32];
2003-361827 [34]; 2003-370772 [35]; 2003-370773 [35]; 2003-456180 [43];
2003-531377 [50]; 2003-554600 [52]; 2003-644797 [61]; 2003-658676 [62];
2003-669970 [63]; 2003-670893 [63]; 2003-677996 [64]; 2003-720863 [68];
2003-720864 [68]
AB US2001038862 A UPAB: 20031022
NOVELTY - A composition comprises an aqueous formulation of: a peptidyl
drug (I), a hydroxide-releasing agent (II), and a carrier (III). (II) enhances the flux of (I) through the body surface.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a system
for topical or transdermal administration of (I) comprising: at least one reservoir containing (I) and (II); a device for maintaining the system in drug and enhancer transmitting relationship to the body surface; and a backing layer. The backing layer serves as the outer surface of the system
during use.
ACTIVITY - Analgesic.
MECHANISM OF ACTION - ***Interferon*** -therapy.
USE - For the delivery of peptidyl drug through a body surface. The peptidyl drug are useful in variety of diseases e.g. pain.
ADVANTAGE - The composition is substantially free of additional permeation enhancer and organic solvents. The formulation increases the
rate at which an active agent permeates the skin, and does not result in sensitization (II) are highly effective
toxicity.
Dwg.0/0

L11 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:851194 CAPLUS
DOCUMENT NUMBER: 136:2090
TITLE: Methods for refolding of growth hormone supergene family proteins containing free cysteine residues
INVENTOR(S): Rosendahl, Mary S.; Cox, George N.; Doherty, Daniel H.
PATENT ASSIGNEE(S): Bolder Biotechnology, Inc., USA
SOURCE: PCT Int. Appl., 110 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087925	A2	20011122	WO 2001-US16088	20010516
WO 2001087925	A3	20020801		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1284987 A2 20030226 EP 2001-941504 20010516
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.: US 2000-204617P P 20000516
WO 2001-US16088 W 20010516

AB The present invention relates to novel methods for making and refolding
insol. or aggregated proteins having free cysteines in which a host cell expressing the protein is exposed to a cysteine blocking agent. Blocking
of free cysteines prevents the crosslinking of proteins into large insol. aggregates, keeping them in soln. and simplifying purifn. and increasing the yield of the biol. activity. The sol., refolded proteins produced by the novel methods can then be modified to increase their effectiveness. Such modifications include attaching a PEG moiety to form PEGylated proteins. The PEGylated proteins of the investigation include recombinant
cysteine variants of members of the growth hormone supergene family such
as: growth hormone, granulocyte colony-stimulating factor, granulocyte macrophage colony-stimulating factor, and .alpha.- ***interferon*** .

L11 ANSWER 6 OF 9 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 2
ACCESSION NUMBER: 2000-096723 [08] WPIDS
DOC. NO. CPI: C2000-028026
TITLE: ***Inhibiting*** ceramide-mediated apoptosis of mammalian cells.
DERWENT CLASS: B04
INVENTOR(S): LEVI, M; SHIMABUKURO, M; UNGER, R H; ZHOU, Y
PATENT ASSIGNEE(S): (TEXA) UNIV TEXAS SYSTEM
COUNTRY COUNT: 84
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9944598	A2	19990910	(200008)*	EN	77

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD
GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV
MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT
UA UG US UZ VN YU ZW
AU 9928931 A 19990920 (200008)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9944598	A2	WO 1999-US4730	19990303
AU 9928931	A	AU 1999-28931	19990303

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 9928931	A Based on	WO 9944598

PRIORITY APPLN. INFO: US 1998-76818P 19980303
AN 2000-096723 [08] WPIDS
AB WO 9944598 A UPAB: 20000215
NOVELTY - A method of ***inhibiting*** ceramide-mediated apoptosis in
reduces ceramide levels in the cell.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a method of treating a subject for beta cell destruction comprising contacting said subject with an agent that reduces levels of ceramide in the cells of said subject as compared to the untreated ceramide level, wherein reduction in ceramide levels protects cells of th subject against ceramide-induced lipotoxicity;
(2) a method for preventing non-insulin dependent diabetes mellitus (NIDDM) in a subject, comprises:
(a) identifying a subject at risk of diabetes mellitus; and
(b) providing to the subject a composition comprising an agent that reduces levels of ceramide in the cells of said subject as compared to th untreated ceramide level, wherein the reduction in ceramide level protects
cells against ceramide-mediated apoptosis of beta -cells;
(3) a method for ***inhibiting*** ceramide-mediated lipotoxicity of a target cell comprising blocking ceramide production or accumulation
in the cell; and
(4) a method of preventing non-insulin dependent diabetes mellitus (NIDDM) in a subject comprising blocking ceramide production in a pancreatic beta cell in the subject, wherein blocking ceramide production
is accomplished by administering to the cell an amount of an inhibitory agent sufficient to protect the cell from ceramide-mediated lipotoxicity.
USE - The process is used to treat a pathologic condition selected from insulin-dependent diabetes mellitus (IDDM)insulin-independent diabetes mellitus (NIDDM) and obesity (all claimed).
ADVANTAGE - The composition is useful for alleviating the deleterious
effects of beta -cell dysfunction and beta -cell destruction caused by lipotoxicity.
Dwg.0/6

L11 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1996:113481 CAPLUS
DOCUMENT NUMBER: 124:137837
TITLE: Host cells transformed with fusion protein gene and method for screening test samples with receptor-ligand interactions or peptide-binding activities
INVENTOR(S): Young, Kathleen H.; Ozenberger, Bradley A.
PATENT ASSIGNEE(S): American Cyanamid Co., USA
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9534646	A1	19951221	WO 1995-US6895	19950531

W: AM, AU, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IS, JP, KG,
KP, KR, KZ, LK, LR, LT, LV, MD, MG, MN, MX, NO, NZ, PL, RO, RU,
SG, SI, SK, TJ, TM, TT, UA, UG, UZ, VN
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT,
LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE,
SN, TD, TG

US 5989808	A	19991123	US 1994-259609	19940614
CA 2195083	AA	19951221	CA 1995-2195083	19950531
AU 9526066	A1	19960105	AU 1995-26066	19950531
AU 706173	B2	19990610		
EP 765389	A1	19970402	EP 1995-920689	19950531
EP 765389	B1	20030716		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE

RU 2208646	C2	20030720	RU 1997-100776	19950531
AT 245189	E	20030815	AT 1995-920689	19950531
ZA 9504892	A	19960130	ZA 1995-4892	19950613
LT 4230	B	19971027	LT 1997-4	19970113
LV 11906	B	19980620	LV 1997-4	19970214
US 6251602	B1	20010626	US 1999-263944	19990308
US 6284519	B1	20010904	US 1999-305483	19990506

PRIORITY APPLN. INFO.: US 1994-259609 A 19940614
WO 1995-US6895 W 19950531

AB This invention relates to novel modified host cells which express heterologous fused proteins and methods of screening for test samples having peptide-binding activity; wherein the modified host cell comprises:
(a) a gene sequence encoding a heterologous fusion protein; said fusion protein comprising a first peptide of a peptide binding pair, or segment of said first peptide, which is joined to either a DNA binding domain or its corresponding transcriptional activation domain of a transcriptional activation protein; (b) a gene sequence encoding a heterologous fusion protein, said fusion protein comprising a second peptide of the peptide binding pair in (a), or a segment thereof, fused to either a DNA binding domain or its corresponding transcriptional activation domain, whichever
one is not employed in (a); (c) a reporter gene operatively assocd. with the transcriptional activation protein, or a portion thereof; (d) optionally, a deletion or mutation in the chromosomal DNA of the host cell
for the transcriptional activation protein if present in the selected host cell.

L11 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1993:227525 CAPLUS
DOCUMENT NUMBER: 118:227525
TITLE: Ecdysteroid dependent regulation of genes in mammalian cells
INVENTOR(S): Godowski, Paul J.

SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9303162	A1	19930218	WO 1992-US6391	19920803
W: JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, SE				
EP 598011	A1	19940525	EP 1992-917507	19920803
EP 598011	B1	19981111		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, SE				
JP 07501928	T2	19950302	JP 1992-503762	19920803
AT 173295	E	19981115	AT 1992-917507	19920803
ES 2123569	T3	19990116	ES 1992-917507	19920803

PRIORITY APPLN. INFO.: US 1991-742127 19910808
WO 1992-US6391 19920803

AB A method of inducing gene expression in a mammalian cell comprises contacting an ecdysteroid receptor with an ecdysteroid within a mammalian cell contg. a gene linked to an ecdysteroid response element. The Drosophila ecdysteroid receptor cDNA was cloned and sequenced. Human 293 cells were cotransfected with a vector contg. this cDNA and a vector in which the chloramphenicol acetyltransferase (CAT) gene was linked to 4 ecdysone response elements. Cat activity was stimulated in cell exts. incubated with muristerone A, but not with .alpha.- or 20-hydroxy-ecdysone or polypodine B, nor with dexamethasone, thyroid hormone, retinoic acid, etc.

L11 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1993:226597 CAPLUS
DOCUMENT NUMBER: 118:226597
TITLE: Selecting agonists and antagonists for growth hormone-like ligands which form ternary complexes with receptors
INVENTOR(S): Cunningham, Brian C.; Devos, Abraham M.; Mulkerrin, Michael G.; Ultsch, Mark; Wells, James
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 77 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9221029	A1	19921126	WO 1992-US3743	19920506
W: JP, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
EP 586549	A1	19940316	EP 1992-912746	19920506
EP 586549	B1	20000920		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE				
JP 06507715	T2	19940901	JP 1993-500070	19920506
JP 3417558	B2	20030616		
AT 196548	E	20001015	AT 1992-912746	19920506
JP 2001270837	A2	20011002	JP 2001-54464	19920506
JP 2003159061	A2	20030603	JP 2002-271918	19920506
US 5506107	A	19960409	US 1993-122548	19930929
US 6429186	B1	20020806	US 1994-308879	19940919

PRIORITY APPLN. INFO.: US 1991-698753 A 19910510
US 1992-864120 A2 19920406
JP 1993-500070 A3 19920506
WO 1992-US3743 W 19920506
US 1993-20327 B1 19930219

AB Growth hormones (GHs), and the class of conformational ligands to which they belong [including erythropoietin, placental ***lactogen*** , prolactin, .alpha.- and .beta.- ***interferons*** , colony-stimulating factors (CSFs), and interleukins 2, 3, 4, 6, and 7], bind sequentially through 2 different receptor-binding sites to 2 receptor mols. to form a 1:2 complex. Formation of such a complex is useful as a criterion for whether a candidate compd. is a ligand agonist or antagonist. The candidate compd. may be a (recombinant) ligand mol. with an altered amino acid sequence whose affinity for the receptor at one or both binding sites is altered. A monoclonal antibody to glycosylated GH receptor was identified which induced body wt. gain in hypophysectomized rats. A cell proliferation assay for GH agonists was developed which used granulocyte-CSF (G-CSF)-responsive FDC-P1 cells transfected with a vector contg. hybrid cDNA for the GH-binding domain of the GH receptor and part of the G-CSF receptor lacking the G-CSF-binding domain.